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Notes on the Coronary Arteries.*

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One hundred years ago all of cardiac pathology was chaos. Corvisart had not revived the discovery of Auenbrugger, and enriched percussion with the results of his own indefatigable observations at the bedside and on the post-mortem table. Laennec had not, accidentally and from his keen sense of decorum, made the immortal discovery that soon revolutionized medical diagnosis. As Corvisart says, many so-called physicians of that day "looked on attempts at an exact diagnosis in heart-disease as useless, because those diseases are incurable." With neat sarcasm he says, in reference to such men, that "art is short, experience silent, and judgment weak."

Yet post-mortem observations on diseases of the coronary, and of the heart-muscle in consequence, are among the oldest in medical literature. Harvey himself (Second Disquisition addressed to Riolan, Jr.,) has recorded a case that must have been one of atheroma of a coronary artery. "Sir Robert Darcy, when he reached about the middle period of life, made frequent complaints of a certain distressing pain in the chest, especially in the night season; so that dreading at one time syncope, at another suffocation in his attacks, he led an unquiet and anxious life. By-and-by he became cachectic and dropsical, and, finally, grievously distressed, he died in one of his paroxysms. In the body we found the wall of the left ventricle ruptured, having a rent in it of a size sufficient to admit any one of my fingers, although the wall itself appeared sufficiently thick and strong." The explanation Harvey gave, that "the laceration had apparently been caused by an impediment to the passage of the blood from the left ventricle into the arteries," is evidently insufficient, but points strongly to aortic disease, in which the coronaries are often affected.

At the end of the last century a few, at least, of the choicer spirits of medicine had a knowledge of myocardial disease by no means inconsiderable, and far in advance of their knowledge of valvular disease. The most illustrious example of this is furnished by the oft-cited case of John Hunter. The acute Jenner correctly diagnosed the calcification of the coronary arteries and referred to them the anginal attacks of his friend and teacher. Parry, too, in several cases was able to make similar predictions, verified post-mortem. On the whole, such cases were not common enough to soon make an impression on the profession as a whole, and it is not surprising that later writers allowed the subject to escape them.

Soon, too, the epoch-making discovery of mediate auscultation engrossed the attention and diverted the minds of physicians especially toward the exam-

*Read, with demonstration of specimens, before the Alumni Association of the Medical Department of the University of Buffalo, May 5, 1896.

ination of the valves of the heart, and away from the muscle itself. The reason for this is not far to seek. It depends on a phase of human nature which shows itself in connection with every new diagnostic method. Auscultation seemed at first to offer a short cut to treatment. The study of physical signs was but too likely to lead to a physical conception of cardiac disease, and the more recondite physiological processes were neglected. To be sure there were exceptions, even during the worst period. Stokes insisted on the importance of a thorough knowledge of the heart-muscle in the treatment of its diseases, but it was not until long after him that the truth was generally received. This came about in various ways. For one reason, it seemed to an increasing number that the sounds and murmurs of the heart did not tell enough. The study of pathological anatomy in more exact methods than were available in the days of Harvey and Morgagni had a rich harvest, and the work of Weigert, Huber and Ziegler led the way for a host of others. The importance of the coronary arteries as a cause of sudden death, examples of which were published in considerable numbers, became generally known, and this led in time to an interest in other consequences of disease in those vessels. From an early period physiologists and pathologists investigated the subject experimentally, with great advantage. It is especially from the work of Dr. W. T. Porter, who was able to bring to the subject not only a clear conception of the problems involved, but also great technical skill, that some of the most intricate points have been cleared up, and the peculiarities of various cases explained. (W. T. Porter, *The Journal of Physiology*, 1893, xv., pp. 121–138. *The Journal of Experimental Medicine*, vol 1, p 46).

Nor have clinicians been idle. The classical paper of Leyden (*Zeitschrift für klin. Med.*, Bd. vii.) and those of Nothnagel, Riegel and Fränkel, and the extensive work of Huchard, have done much to further a knowledge of the subject and led to countless contributions of value. Many text-books give an adequate summary of the matter, but others appear not to recognize its importance. That recognition in general has been slow is partly due, no doubt, to a peculiarity of the disease or diseases in question. From the nature of the cases hospital physicians see but a part of their course, and that not always the most important for a successful diagnosis. It must, therefore, remain for those physicians in general practice who have an oversight of patients for long periods, and who at the same time have the sort of scientific zeal that influenced a Jenner to give the final touches to the pictures of coronary disease.

It is not necessary to enter into details concerning the anatomy of the coronary arteries. It is enough to recall certain cardinal features. Various parts of the heart-muscle are nourished by branches of the coronaries, the distribution of which is fairly regular. The left coronary supplies the greater part of the left ventricle, and is especially important because it is found diseased more frequently than the right. The occasional occurrence of supplementary coronary arteries, as they are called, really anomalous origins of branches from the sinuses of Valsalva, is of no clinical importance.

The question as to the anastomosis of the two coronary arteries has led to much controversy. As a rule, there is no arterial anastomosis, though rare cases may have been observed in which these actually existed. The capillary anastomosis, however, may become enlarged as the result of disease, a fact of great importance in certain cases. The rapidity with which an obstruction forms is, therefore, decisive in regard to the question of collateral circulation.

The most frequent disease affecting the coronary arteries is arteriosclerosis in its various forms. This may be part of a wide-spread arterial dis-

ease, or it may be localized in the coronaries, or in the beginning of the aorta, around the orifices of the former.

The causes of the great frequency of coronary sclerosis have been much discussed. In addition to causes operating on other vessels, it may be that the stress in the arteries of the heart, secondarily their peculiarities of structure, especially the unusually thick adventitia, have a determining influence.

Changes of slight degree are common in the coronaries of persons beyond middle life without apparently producing symptoms, and even without greatly affecting the heart-muscle. In others, fatty degeneration or fibroid change occur in varying degree, and often produce symptoms of considerable importance. In others the results are more immediate and striking. Instead of attempting to describe them formally, I shall recite some case-histories illustrating various forms.

Case 1.—Angina Pectoris; sudden death in first paroxysm. Sclerosis of the coronary arteries; rupture of an atheromatous focus in the descending branch of the left coronary artery; multiple embolism of the heart.

A negro drayman of fifty years, a man of unusually powerful physique, with a history of perfect health, was seized in the night with pain in the heart region and a sense of suffocation. He was seen by Dr. H. A. West, who found no special symptoms other than those mentioned. Morphine temporarily relieved the pain, but in about two hours after the onset the patient suddenly died. I made an autopsy six hours after death, and finding no marked evidence of disease in any other organ, took the heart unopened to my laboratory for careful examination. This was made by cutting the organ in slices, parallel to the auriculo-ventricular septum, and examining the vessels in each slice separately. This proved to be a useful method, though destroying the heart as a specimen.

The heart was slightly enlarged, both ventricles dilated, only moderately hypertrophied. The chambers contained a small quantity of uncoagulated blood. The muscle was soft, dry, pale-brown, with small yellow spots of pin-point size. The left papillary muscle was fibroid and calcified at the tips. All the valves normal. The aorta, smooth as far as the arch, was atheromatous and calcified from there to the bifurcation. The branches of the aorta were all free from atheroma except the coronaries. The latter were affected in various degrees in their whole extent, being calcified, partially occluded or dilated. In the descending branch of the left, just below the origin, was a ruptured atheromatous abscess, eight mm. long, extending two thirds around the dilated artery. The edges were overhanging, the floor uneven and ragged, presenting all the appearances of a recent rupture. This proved to be the case. On opening up the sections of the branches below the rupture they were found to contain a little blood with characteristic atheromatous material, such as cholesterin plates, blood-pigment in crystals and masses, large cells with highly refracting granules, and amorphous and granular debris. The left lateral branch of the artery just below the rupture was obstructed by an atheromatous nodule in its wall, but all the other branches contained debris as far as they could be traced.

Fresh frozen sections of the heart showed fibroid degeneration, with many small areas of fatty and albuminous degeneration. Hardened sections showed: endarteritis nodosa; extreme congestion of capillaries; brown atrophy of muscle cells; swelling of nuclei; fibroid degeneration; fragmentation of the heart-muscle. In many cases the spaces between the ends of the ruptured fibres are filled with blood, making it highly probable that the process was ante-mortem.

This case requires little comment. Notwithstanding the patient's state-

ment as to his previous health we may be confident that an examination of the heart under favorable conditions would have revealed evidences of myocardial disease. The cause of the embolism was a rare one. Most cases of coronary embolism are due to bits of thrombi or vegetations from the aorta or heart. In the case of the sculptor Thorwaldsen, however, who died suddenly in the theater, the condition was precisely the same (*Virchow's Archiv.* Bd. 25, p. 315).

Such cases are quite in accord with the results of experiments on animals, viz.: that the *sudden* closure of a large artery, almost always, that of several smaller ones, usually causes total arrest of the heart's action and death within a comparatively short time. Such closure is most frequently due to a thrombus, as occurred in:

CASE II.—Angina pectoris; sudden death in an attack. Thrombus in atheromatous right coronary artery.

Mrs. M., aged sixty; laparotomy and removal of both ovaries for cystic disease. Examination before the operation showed no evidence of heart disease, and the patient gave no history of cardiac symptoms. Five days after the operation, when healing seemed to be progressing nicely, the patient complained of a sudden pain in the heart region, shooting into the left shoulder and arm, with a feeling of suffocation, but without objective dyspnea. She then recalled similar attacks, not so severe, at long intervals. Inhalations of amyl nitrite and an injection of morphine gave only slight relief, and the patient died soon after the attack began.

On examination I found the heart large, weighing eleven and three-quarter ounces, with an excess of sub-epicardial fat. (The patient was not fat). Both ventricles contained ante-mortem clots. The endocardium and valves were normal. The wall of the left ventricle measured twenty mm. at the middle, the muscle was brown, with numerous fibroid patches, especially in the bases and tips of the papillary muscles. The aorta showed numerous large and thick atheromatous plaques. The left coronary artery was atheromatous, the lumen contracted in various parts, but not markedly obstructed in any of the larger branches. The right coronary was also atheromatous. Two cm. below the orifice the lumen suddenly widened from eight to thirteen mm. in circumference, the dilated part being thirty-five mm. long. The walls there were rough, thin, calcareous. Adherent to the wall was a mixed white and red thrombus.

This was, doubtless, the cause of the sudden death, and the case is interesting chiefly because the right coronary is rarely the seat of a fatal complication. Here, again, it would seem that a critical examination of the heart during life would have revealed the hypertrophy, as well as some alteration in the sounds or the pulse sufficient to make a diagnosis of myocardial degeneration. Such findings are not grounds for preventing an operation when it is needed, and the fatal termination must be looked on as purely accidental.

Very different from such cases are those in which the closure is slowly brought about, though affecting even a main trunk.

CASE III.—Aortic valvular disease with loss of compensation; dilatation of the heart; anginose attacks; sudden death. Aortic stenosis and insufficiency; aortic atheroma; occlusion of the right coronary artery.

F. C., aged thirty-six. Patient has a history of atypical sore on the penis without secondary symptoms; no specific treatment. Later married and wife had one healthy child. Used alcohol in moderation. No history of acute illness indicating possible relation with present disease. In the fall of 1893 noticed sharp pain under the sternum radiating to the back and down the left

arm and into the left hand. This recurred at intervals and has now been constant for six weeks. The pain is worse after exertion and while lying down, especially when lying on the left side. In the latter position the pain is associated with a feeling of suffocation. There is no history of dyspnea, cyanosis, or ascites.

Present condition, August 2, 1894: Slender frame; musculature poor; panniculus scanty; skin pale and sallow, no cyanosis, no edema. Apex-beat visible in the fifth interspace one inch outside the left nipple line; the impulse is heaving; there is no thrill. Pressure over the heart is not painful. Absolute heart dulness begins on the upper border of the fourth rib, extending to the fifth interspace in the nipple line, and to the right parasternal line in the fifth interspace. The first sound at the apex is inaudible; no murmur can be heard there. At the aortic cartilage there is a short, soft systolic murmur, followed at once by a louder and longer blowing murmur. Both murmurs are well heard over the carotids and the diastolic murmur can be heard over the base, down to the left nipple and along the sternum.

Radial pulse 120, regular, moderately full but very quick (water hammer). There is slight pulsation of the peripheral arteries. Liver dulness not enlarged; palpation and percussion of abdomen negative. Urine (twenty-four hours), 500 ccm.; sp g. 1029; reddish-yellow, clear; no albumin, no sugar. The scanty sediment contains a few leucocytes and cylindroids.

Under rest in bed the heart's action became slower and a systolic murmur could be detected at the apex, distinct from the former one, transmitted to the axilla. The pain in the cardiac region continued. It radiated to the back and shoulders; it was not accompanied with subjective or objective dyspnea. Later the pulse became more frequent and smaller. Orthopnea came on. Digitalis had no effect on this. Late in the evening of August 4, two days after admission, the patient complained of nausea and pain in the heart, and died suddenly, two hours after midnight.

The autopsy showed yellowish-red subcutaneous fat; slight excess of fluid in all the serous cavities. Lungs congested; no edema. The heart extended from the right parasternal to the left anterior axillary line. All the cavities were dilated, the right auricle and left ventricle being especially affected. The left ventricle was globular, encroaching on the right, ten cm. long and nine cm. wide. The wall just below the ring was thirteen mm. thick; at the apex seven mm. The muscle was pale, soft; showed a few small fibroid areas. The papillary muscles were long and flattened; showed exquisite fatty degeneration and numerous long and narrow fibroid patches. The mitral orifice permitted three fingers to enter readily. The flaps were normal. The walls of the right ventricle measured 4.5 mm. at the upper part; two mm. at the apex. The tricuspid admitted three fingers. The pulmonary artery was ten cm. in circumference just above the valves; nine just below the division.

The aorta was eight cm. in circumference. The valves were thick, rigid, and retracted close to the wall. From the insertion of the valves to a distance of five cm. above, the surface was wrinkled, and covered with irregular sclerotic plaques.

The right coronary artery was completely occluded at the orifice, being involved in the atheromatous process. The orifice of the left coronary was narrow but free. It was readily opened by fine probe-pointed scissors as far as two cm. beyond the apex, where it ended in four small branches. Just at this point the artery had a circumference of five mm. The branches were from .5 to 1 mm. in diameter and emptied after from two to five mm. into the end of

the right coronary artery. All the branches of the coronaries were smooth, without atheroma. The spleen, liver, kidneys, and intestines showed cyanosis of moderate degree.

The most important feature of this case is the coronary anastomosis after gradual occlusion. This must be a not infrequent process, but the anastomosis is usually not found. I have seen two other cases with chronic occlusion of one coronary. In one no gross anastomosis could be seen, and there was no opportunity for a careful examination. In the other the heart was so decomposed that a satisfactory examination was impossible, but it could at least be said that no large vessels were concerned. In the former case the clinical history was unknown. In the latter, a negro woman of twenty-six years, death occurred while turning over in bed. There was a history of "asthma" and dropsy for six months. The aorta and its valves were extremely atheromatous and calcified, the valves being quite obliterated. The orifices of the coronaries could not be seen. The right was found by passing a probe from below, but the orifice of the left was found to be totally occluded.

The cause of the anginose pain in Case III may have been the coronary disease, but was more probably due, as was supposed during life, to the aortic disease and, toward the end, acute dilatation. That the case was syphilitic can hardly be doubted.

When acute occlusion affects one small branch, or even a number of small branches, without involving enough to cause sudden death by anemia of the heart-muscle, local anemia or infarction occurs, with the condition of myomalacia, as Ziegler called it. This is a not uncommon accident and I could cite a number of examples, but in most of them, in fact in all but one in which a diagnosis was made during life, the symptoms were complicated by disease of the kidneys, lungs or brain. This one case, however, was simpler and therefor worth relating.

Case IV.—Angina pectoris; dyspnea; double hydrothorax; sudden death. Atheroma and obstruction of coronaries; infarction of the heart.

Mr. B., lumber dealer, sixty-four years old, a man of large frame, was never sick until about three months before death. He then began to notice shortness of breadth, especially when walking up hill. A week before death severe pain in the heart-region began. There was no clear history of radiating pain. Soon after this, in rising suddenly, the patient fainted, became pulseless and very dyspneic. Dr. Breakey was called in and made a diagnosis of angina pectoris, prescribing nitro-glycerine and strophanthus. The symptoms continued and I saw the patient a week later. He was then lying propped up in bed, but anxious to get out. There was slight cyanosis; breathing rapid and superficial; pulse eighty, small, quick; no atheroma of the peripheral arteries.

The apex-beat could not be felt. The heart dulness extended from the fourth rib to the fifth interspace, and from the left edge of the sternum to beyond the left parasternal line. As the lungs gave a very tympanitic note, it was supposed that the heart dulness might be masked by over-distended lungs. The sounds over the heart were faint but clear. Over the apex-region a loud double friction-sound was audible. There was a small area of movable dulness in each side, which had been present for two or three days. Loud moist râles obscured the respiratory murmur. The abdomen was considerably distended, the tongue coated; constipation had existed for some time. Repeated examinations of the urine were negative.

The diagnosis was myomalacia following coronary sclerosis, with second-

ary pericarditis. This was based on the history of increasing dyspnea and heart pain, without evidence of disease in lungs or kidneys, or other (valvular) disease of the heart, the history of the acute attack indicating infarction, and the acute onset of pericarditis without other cause.

The same day, against explicit orders and explanations of the danger from exertion of any kind, the patient insisted on being helped out of bed to defecate. While straining at stool he suddenly expired.

Autopsy showed about a pint of clear fluid in each pleural cavity, congestion and edema of the lungs. The heart was enlarged, reaching the left nipple line. The pericardium was adherent over the apex by a thin, greenish fibrinous exudate, easily separated. The pericardium was uniformly reddened and rough.

The surface of the left ventricle, beneath the thin exudate, was mottled red and green in irregular spots. Both ventricles were dilated, the left being 10.3 cm. long, its wall fifteen mm. thick at the upper part, seven mm. thick at the apex. The walls of the right ventricle were five mm. and three mm. thick. at the upper part and apex respectively. All the chambers contained soft, dark-red clots.

The aorta, seven cm. in circumference, showed a few atheromatous areas around the orifices of the coronaries, but the latter were not obstructed.

Just below the orifice the left coronary artery became extremely atheromatous, nodules of two to three mm. in diameter, in the walls, obstructing the lumen. The descending branch was narrowed, calcified, and about the middle of the anterior wall was obstructed by a red thrombus. Below this the artery was smooth. From the level of the thrombus the muscle was dry, yellow and red in irregular areas, and tore easily. Adherent to the endocardium over this part was a thin mixed thrombus.

The circumflex branch was nodular, but its lumen was free as far as the first branch, 2.5 cm. from its origin. Here it was completely obstructed by nodular arteritis for a distance of three mm. Beyond this, the lumen of the circumflex proper was free, but the next large descending branch was also totally obstructed. The wall of the left ventricle from this point, *i. e.*, from the anterior papillary muscle, to the septum, the posterior part of which was involved, and from near the ring to the apex, was the seat of a recent infarction. Only a thin layer, under the epicardium, from one to two mm. in thickness, was not necrosed, and it was red, swollen, the fibres cloudy and granular. The non-infarcted parts of the heart showed brown atrophy, fibroid degeneration and fatty change.

In this case the relation of the coronary sclerosis to the gradually developing dyspnea, and of the infarction to the acute attack a week before death is clear. The case illustrates also the fact that a heart extensively necrosed may continue to act for some time fairly well, if exposed to no sudden strain, thus explaining the early stages of those cases in which large fibrous areas are found *post-mortem* in the heart. It is also a fine example of atheroma limited almost entirely to the coronary vessels. Usually there are other evidences, but in rare cases only one set of vessels is involved, a point brought out prominently in the diagnosis of this case.

I have not intended to speak in detail of the symptoms of coronary artery disease, nor even to touch on the important points of prognosis and treatment. If I have been able to interest you in the subject in such a way that new light will be thrown upon it, I shall have additional cause for congratulating myself on being with you to-day.